Blood Lead Levels Among Children in Hawaii

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The objectives of this study were to estimate blood lead levels of children under 6 years of age in the State of Hawaii, to identify high-risk populations, and to decide what kind of blood lead screening program is needed in Hawaii. Children from Oahu and Maui were recruited in medical care facilities and 6 preschools. Informed consent for a blood lead test was obtained from the parents. A questionnaire was administered whenever possible; participation/response rates were quite low, varying between 15% and 50%.

The mean blood lead level for the 389 study participants was 4.5µg/dL. Twelve children (3% of children tested) had a level above 9µg/dL. No child was found to have a level above 14µg/dL. Blood lead levels varied by age, geographic area, occupational factors, laboratory that performed the analysis, and type of phlebotomy (capillary versus venous) but not by sex, ethnicity or age of home. Follow-up investigations in the 12 homes of the children with levels above 9/lg/dL were performed and in 3 homes a possible source of lead exposure was found.

Introduction

Despite the low participation rate, the study appears to be fairly representative of Hawaii's children since it included children from different geographic areas and housing types, with varying socioeconomic and educational backgrounds. There is no reason to think that the true distribution of blood lead levels among children in Hawaii differs drastically from the results in this study.

Education of the public and alerting the physicians are needed to reach families whose children are at risk for lead exposure. Children exposed to any one risk factor (living in an old home with peeling paint, parents' occupational exposure to lead, water catchment system) should receive a blood lead test. Mandatory lead testing of all children in Hawaii would not be cost-effective and is not recommended.

Until recently, blood lead levels that did not cause symptoms of lead poisoning, namely anemia, abdominal pain, paralysis and encephalopathy, were considered safe. However, growing evidence indicates that low levels of blood lead have subtle adverse effects in children[5,6]. Neurobehavioral effects are of special concern during the fetal period and the early years of life because the neurologic system develops rapidly during that time. Lead's neurotoxic effects at relatively low exposure levels include impaired mental or motor development, decreased intelligence, learning disabilities, impairment of visual-motor functioning, poor perceptual integration and poor memory, hyperactive behavior and alteration of hearing thresholds. The damage to the neurologic system caused by lead is considered irreversible.

Lead has no essential function in the human body. Studies among remote populations provide evidence that there is no "normal" blood lead level in the sense of a natural background level[1,7]; human blood lead levels are caused entirely by the products of industry and the resulting dissemination of lead into the environment. In 1970, a level of 60/g/dL was defined as "undue lead absorption." One year later this was lowered to 40/g/dL. In 1975, the Centers for Disease Control (CDC) changed the limit to 301g/dL and in 1985 to 25/Jg/dL[10]. In October 1991, the CDC published a recommendation to lower the level of "undue lead absorption" to below 10/g/dL[11]. Levels in the border zone of 10 to 14/Jg/dL should be rescreened and should trigger a community public health investigation. The steady lowering of the accepted threshold for lead's toxic effects can be interpreted as evidence that there is no intrinsic threshold for lead and thus presents a "continuum of toxicity."

Average blood lead levels in the United States decreased from 14.6 to 9.2 /Jg/dL from 1976 to 1980, and has possibly decreased since then[11]. This change has been attributed to the reduction of organic lead additives in gasoline during the same time period. However, other possible sources for lead exposure remain: Lead-based paint, lead in soil, foods, drinking water, air, as well as occupational exposure.

The main source of exposure to lead among lead-poisoned children in urban areas is lead-based paint. Children may ingest lead directly from paint chips, but an important route of exposure is the normal mouthing of hands or objects such as toys, resulting in the ingestion of small amounts of lead-paint-contaminated house dust and soil. Children living in deteriorating housing built before 1950 are at high risk for excessive exposure to lead via this route. The lead content of paint used during that period varied; with some, particularly in earlier years, containing as much as 50% lead by dry weight. The 1987 Housing Act established an allowable level of not more than 0.5% for paint used in public and Indian housing and on Indian reservations.

The Federal Government has made lead abatement and the prevention of childhood lead poisoning a priority. The U.S. Department of Health and Human Services (DHHS) published a Strategic Plan for the Elimination of Childhood Lead Poisoning in February 1991[12]. Several environmental organizations, such as the Environmental Defense Fund[2] and the Alliance to End Childhood Lead Poisoning, concentrate their efforts on this issue also.

The most recent data on blood lead levels available for the State of Hawaii come from a study on the Big Island (unpublished data) conducted jointly by the CDC and the Hawaii State Department of Health (DoH). In 1988, CDC-DoH investigated water catchment systems and offered testing for lead to families consuming drinking water from rainwater catchment. From the participating 93 children under age five, 12 (13%) had blood lead levels above 10µg/dL and 6 (6.5%) above 15µg/dL. The mean blood lead level was 6.4µg/dL. In 1973 a small study compared blood lead levels of children in Hawaii and New Jersey[4]. The mean blood lead level in Hawaii was 17µg/dL at the time. Other studies performed in the state used zinc protoporphyrin (ZPP) as a screening test which does not identify children with blood lead levels below 25µg/dL[9]. Nevertheless, the data provided evidence that very few children in Hawaii experienced severe lead toxicity.

Our present study was initiated when a committee within the DoH realized data on blood lead levels were lacking at a

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time when new CDC recommendations to lower intervention levels for lead were imminent. The objectives of our study were to estimate blood lead levels of children under 6 years of age in Hawaii, to identify high-risk populations, and to decide what kind of blood lead screening program is needed in Hawaii.

Methods

Children from Oahu and Maui were included in the study and were recruited either in a medical facility or in a day-care center (Table 1). Budget constraints and logistical problems made it impossible to include the Big Island and Kauai. Blood samples were collected between March and August 1992. In the participating clinics, research assistants talked to parents of children <6-years old, preferably to those who were scheduled to have blood taken for other reasons. Some older children were included on parents' requests. After explaining the purpose of the study, the research assistant obtained written informed consent for the blood lead test and administered a short questionnaire. Blood was taken in the lab after the child had been given a health check by the doctor. Table 1 shows estimated acceptance rates. A high percentage of parents refused to have their child tested and another smaller percentage failed to take the child to the lab even though they had signed the consent form. Physicians at the Waianae Coast Comprehensive Health Center, at Maui Medical Group, and in private practice recruited their own patients for the study and no information on participation rate is available from these sources.

The preschools were selected to cover different areas on Oahu. Not all preschools that we approached were willing to participate; one organization refused because of legal concerns. In the cooperating schools, we sent a letter to the parents of the preschool children asking them to sign the consent form and to send it back to school. Depending on the school, between 15% and 30% of the parents consented. The highest acceptance rate was achieved in Kalihi's Headstart and Zero-to-Three Program where we had the opportunity to meet with some of the mothers and to explain the significance of high blood lead levels to them. The blood from the pre-schoolers was drawn at the school on a designated day.

The blood lead analysis were performed by 3 different laboratories. The choice of a particular lab for a sample was dictated by practical considerations. One of the labs (Kaulson) is located on the East Coast. Two are located in Hawaii (SmithKline and Diagnostic Laboratory Services) but these also send their samples to the Mainland for analysis. At this time no laboratory in Hawaii performs blood lead analysis routinely. All 3 labs participate in regular quality assurance programs. The measurement error for the atomic absorption spectrometry method is approximately +2µg/dL. To avoid contamination, venous blood was drawn whenever possible; 237(61%) of all samples were venous samples. However, some of the clinics preferred to take capillary samples, and they were instructed on how to clean the finger appropriately.

Interviews were conducted over the phone in those families who had not been recruited by one of our research assistants. The questionnaire contained questions about the child's behavior and health, the child's home, risk factors for lead poisoning such as parents' occupation and hobbies, ceramic dishes, education and income level (Appendix). Test results were returned to the health care providers for children recruited in clinics and to the parents for children recruited in preschools. Parents of children with blood lead levels of 10/Jg/dL and above were contacted and offered a home visit to check for lead in paint and other potential sources. A commercial kit for instant environmental lead testing was used (Leadcheck™).

The zip code information was aggregated into larger geographic areas with the goal of calculating mean levels of blood lead from at least 10 samples per area. To get a rough idea in which areas sampling proportions were higher or lower than in the entire study, a sampling index was calculated. The number of

TABLE 1: Locations of Sample Collection and Participation Rate

	Location	Number Tested	Percent Participation	Mean blood Lead (µg/dL
CLINICS	**All**	**445**	**?**	**4.1**
	DOH-Kauai	166	?	3.4
	Kaiser Moanalua	70	50%	4.7
	Kaiser Honolulu	67	25%	5.2
	Kapiolani	61	20%	4.8
	Waianae Coast	12	?	5.3
	Maui Medical	49	?	2,9
	Other	20	?	5.6
PRESCHOOLS	**All**	**124**	**23%**	**4.0**
	PTA (Kalihi)	52	30%	4.7
	Olivet Baptist	22	25%	3.5
	HCC-Waipahu	15	20%	3.2
	HCC-Kailua	17	20%	3.5
	HCC-Salt Lake	9	15%	2.0
	Kamehameha/Waimanalo	9	20%	5.6

FIGURE 1. DISTRIBUTION OF 569 BLOOD LEAD LEVELS IN CHILDREN

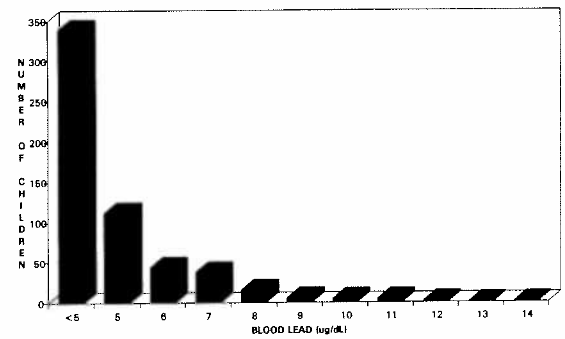

TABLE 2: Characteristics of Study Population

		Number	Percent	Census90
SEX	male	289	51	—
	female	280	49	—
AGE	<1 year	35	6	—
	1 year	119	21	—
	2 years	90	16	—
	3 years	117	21	—
	4 years	137	24	—
	>5 years	15	3	—
RESIDENCE	Oahu	353	62	—
	Kauai	166	29	—
	Maui	35	6	—
	Big Island	1	<1	—
ETHNICITY (n=334)	Hawaiian	105	31	—
	Mixed	83	25	—
	Samoan	35	10	—
	Filipino	24	7	—
	Caucasian	35	10	—
	Japanese	21	6	—
	Chinese	7	2	—
	Black	6	2	—
	Other	18	5	—
FAMILY INCOME (n=314)	<$15,000	96	31	10
	$15-30,000	92	29	21
	>$30,000	126	40	69
EDUCATION OF PARENT (n=326)	<12 years	24	7	20
	High School	115	35	29
	College	149	46	44
	Graduates	38	12	7
AGE OF HOME (n=330)	<15 years	100	30	21
	15-30 years	125	38	52
	>30 years	105	32	27

tests performed per geographic area was divided by the number of births in 1991 for the same geographic area. The resulting figure for each area was divided by the overall ratio, ie 389 tests by 19,880 annual births. This index is greater than one if proportionately more children were tested in a particular area than across the State and less than one if proportionately fewer children were tested than across the State. The study made an effort to include many children from areas that were considered high-risk because of old housing, such as Waimanalo, Kalihi and Waianae.

All data were entered into a spreadsheet. The statistical analysis was performed with the help of SAS, using standard procedures for calculating prevalence rates, chi-squares, and T-tests.

Results

Altogether, 389 children were tested for lead. Characteristics of the study population are listed in Table 2. The mean blood lead level in this study was 4.5µg/dL with a standard error of 0.11. That means the population mean for children in Hawaii can be expected to lie between 4.3µg/dL and 4.7µg/dL, assuming that the study participants are representative of the population. The overall distribution of blood lead levels (Figure 1) shows that over 50% of all lead levels were < 5µg/dL. Twelve children had a level above 9µg/dL (3% of all children tested). No child was found to have a level above 14µg/dL.

FIGURE 2. DISTRIBUTION OF BLOOD LEAD LEVELS BY AGE

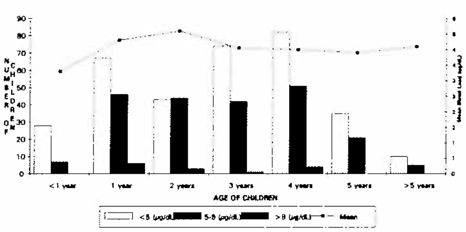

Figure 2 illustrates the distribution of blood lead levels by age. As expected, the younger children had a somewhat higher proportion of levels above 5µg/dL. The mean blood lead level differs significantly ($p=0.04$) among age groups. Infants under one year had the lowest mean level with 3.8µg/dL, whereas 1- and 2-year-old children had slightly higher mean levels (4.9µg/dL and 5.1µg/dL respectively). This difference is significant ($p=0.04$).

The sex of the child was not associated with the blood lead level; the mean was 4.6/Jg/dL and 4.4g/dL for boys and girls respectively. Information on ethnicity was available in only 278 children. Among those, no statistically significant difference in lead levels was found (Figure 3). Filipino and Japanese children were underrepresented in the study.

Mean blood lead levels by geographic area (Table 3) range between 3.2µg/dL on Maui and 5.6µg/dL in Aiea. Considering the measurement error of the lab method, this range is quite narrow. The sampling index indicates areas with a high proportion of samples, especially Waimanalo, Kalihi, Manoa and Moiliili/McCully, whereas Pearl City, Salt Lake, Waianae Coast, Windward Oahu and the north shore were less well represented. The smaller the standard error of the mean for each area, the more stable the mean blood lead level for this area.

The mean blood lead levels differed significantly among the 3 laboratories: Kaulson 5.0µg/dL, SmithKline 4.5µg/dL, and Diagnostic Lab Services 3.4µg/dL. The mean blood lead level for capillary samples was 4.9µg/dL which statistically is significantly higher than the mean of 4.2µg/dL for venous samples. The

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difference between the labs persists after controlling for age and type of phlebotomy (venous versus capillary). Figure 4 illustrates the complex relationship between age of child, type of phlebotomy, and laboratory.

Of the 389 in the study, 284 (73%) included a parental interview. The mean blood level of those interviewed was significantly higher than in those not interviewed (4 7µg/dL versus 4.0µg/dL, $p<0.004$). Of all children whose parents were interviewed, only 3 (2.1%) had ever received a lead test before. Of 187 (67%) children, their health was described as excellent or very good. The blood lead levels did not differ significantly based on health status nor by number of symptoms experienced during the previous 3 months. Table 4 lists mean blood lead levels by risk factor. The differences between the means are minute for the most part and only 3 are statistically significant: Putting fingers into mouth, welding as father's occupation, and mother's occupational status as homemaker. Of the 178 children living in homes less than 30 years old, the mean blood lead was 4.6µg/dL, whereas in the 95 children in homes older than 30 years, the level was 4.99. That difference is not statistically significant.

The parents of the 12 children (all in different families) with levels above 9µg/dL were offered follow-up investigations. Five families agreed to an investigation, 5 families could not be contacted, 1 family refused a visit and 1 home had already been checked for lead by the Housing Authority and no lead had been found. In 2 of the 5 homes visited, no leaded paint or other potential source for lead could be identified in or around the home. The babysitter's house could have been a source of lead for 1 of those 2 children. In one home, older layers of paint in the kitchen and outside the house contained lead. Most of it was in a satisfactory state and the parents were advised to take care of the few flaking spots. In another home the paint was new; however, the father, who was occupationally exposed to lead, brought home metal pieces containing lead and the children had actually played with them.

The child with the highest level (14µg/dL) lived in a modern apartment building with latex-based paint. However, several old buildings had been torn down in the neighborhood and dust could have blown into the area where the child played outside. Since some tests had been done on capillary samples, a repeat test was recommended to these 12 families. So far, no family has taken the children to the lab for retesting.

Discussion

The mean blood lead level of 4.5µg/dL and the 3% proportion of levels above 10µg/dL in the 389 children tested is very low in comparison to the Big Island roof catchment study in 1988 and as the national studies. The trend of decreasing levels has been observed across the nation and is attributed to the introduction of unleaded gasoline[11]. We would like to know how many children in Hawaii might have levels above 14µg/dL despite the fact that no child with such a level was found in the study. Assuming that the 389 study participants are representative of all children in Hawaii, the upper limit for a 95% confidence interval for the proportion of children with levels above 14µg/dL can statistically be calculated as 0.0077. That means a 95% probability that not more than 0.77% of all children have blood lead levels above 14µg/dL. In approximately 100,000 children < 6-years old, as many as 770 children in Hawaii could have levels above 14µg/dL. Such a number is considerably lower than that estimated by the Agency of Toxic Substances and Disease Registry[13]. In 1988, they estimated that 31% of all children in Honolulu might have levels above 9µg/dL and that 9% had levels above 14µg/dL.

The major shortcoming in our study was the enormous rate of refusal to undergo a blood lead test. Many parents had never heard about lead. Many of those who had heard about it believed that they did not have a problem. However, the strongest factor in deciding whether or not to participate seemed to be the reluctance of the parent (even among health and public health workers) to have a child undergo a phlebotomy. On the other hand, the children themselves were mostly cooperative and showed few signs of fear when they underwent phlebotomy at the preschools.

If the 389 samples are representative of children in Hawaii, this study shows that lead is not a major health problem for children in the state. There are some indications that most population groups were represented in our study. The proportion of Hawaiians/Part Hawaiians in the study is approximately equal to their proportion among recent births. Recent immigrants were included through the Headstart and Zero-to-Three program in Kalihi. By comparing the income structure of the study families to that in the 1990 census (Table 1), it can be interpreted that a greater proportion of low-income families were included in our study. Educational attainment was slightly higher among study participants than in the general population. The age of the home, as far as it was known, was approximately representative of homes in general. It may still be possible that those children whose parents did not accept a lead test had radically different lead levels than our participants.

Certain geographic areas are under-represented in our study. For the island of Kauai, there is no known reason to indicate that lead

FIGURE 3. DISTRIBUTION OF 334 BLOOD LEAD LEVELS IN CHILDREN BY ETHNIC GROUP

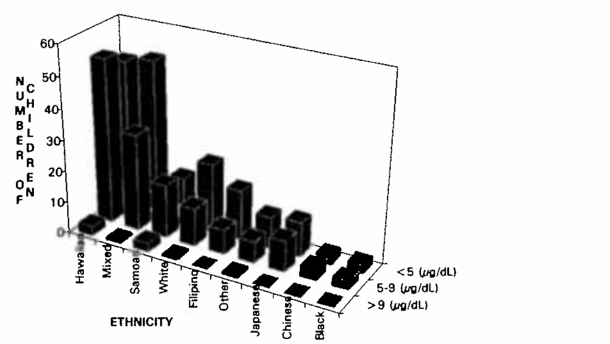

TABLE 3: Mean Blood Lead Levels by Geographic Area

Island	Area	Number Tested	Mean Blood Lead (µg/dL)	Standard Error	Sampling Index*
OAHU	Aiea	11	5.6	1.02	0.6
	Ala Moana/Waikiki	13	5.3	0.71	1.0
	Downtown	13	4.8	0.38	1.7
	East Honolulu	23	4.1	0.22	0.9
	Ewa Beach	23	3.9	0.33	0.7
	Kalihi	76	4.9	0.23	2.6
	Manoa	22	4.6	0.36	1.8
	Moiliili/McCully	19	4.6	0.63	1.6
	Nuuanu	25	5.0	0.35	1.2
	Pearl City	10	4.1	0.53	0.6
	Salt Lake	18	3.6	0.44	0.4
	Wahiawa/Mililani/ North Shore	18	4.8	0.54	0.3
	Waianae	17	5.5	0.59	0.6
	Waimanalo	22	4.4	0.41	3.4
	Waipahu	19	4.8	0.69	0.6
	Windward	19	4.5	0.56	0.4
KAUAI	East Kauai	84	3.2	0.19	5.9
	West Kauai	68	3.4	0.23	7.8
	North Kauai	14	3.8	0.55	2.8
MAUI	All areas	49	2.9	0.29	1.2
UNKNOWN/ OTHER		6	4.2	0.83	n.a.
TOTAL		569	4.1	0.09	1.0

* Index >1 indicates a higher than average number of children were tested.

levels would be drastically different from the rest of the state. For the island of Hawaii, it is known that some water catchment systems contain lead. Data from the 1988 CDC-DoH study showed a mean blood lead level in children under 6 years to be 6.4µg/dL, which was higher than that in the present study. Such a higher value could be a result of the lead in the drinking water from roof catchment systems, where Vog-related acid rain tends to leach lead from nails, solder, or paint and into drinking water.

Measurements of low levels of lead in blood are fraught with many technical problems. Contamination of test tubes and lab equipment may increase the true lead concentration up to 150%[3]. Capillary samples also might be contaminated with lead from a finger. Considering these factors, the actual blood lead levels in Hawaii could be even lower than this study indicates. The lab that used only venous samples and a particular type of vacutainer designed for heavy metal analysis had the lowest blood lead mean (3.4µg/dL). It is unfortunate that not all participating clinics agreed to draw venous samples into blue-top vacutainers.

Information from the questionnaire provided very little explanation about the variation in lead levels among study participants.

FIGURE 4. MEAN BLOOD LEVELS BY AGE, LABORATORY, AND TYPE OF PHLEBOTOMY

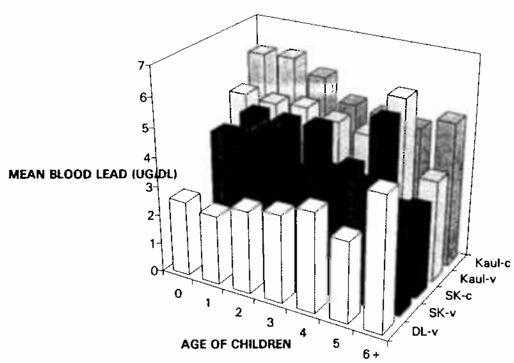

TABLE 4: Blood Lead Means (µg/dL) for Selected Risk Factors		NO		YES	
		Number	Mean	Number	Mean
BEHAVIORAL RISK FACTORS	Chew on toys	192	4.3	144	4.7
	Eat dirt	271	4.4	64	4.7
	Pick paint	231	4.4	97	4.7
	Eat paint	309	4.5	18	4.9
	Fingers in mouth	80	4.1	259	***4.6***
	Chew on furnitures	281	4.4	57	4.8
	Use pottery, cans or ceramics for food	175	4.5	161	4.5
RESIDENTIAL RISK FACTORS	Urban neighborhood	181	4.4	139	4.7
	Separate house	122	4.7	150	4.2
	Home older than 30 yrs.	225	4.3	105	***5.0***
	Peeling paint in home	194	4.4	131	4.7
	Recent paint removal	230	4.5	76	6.4
OCCUPATIONAL RISK FACTORS	Battery work	301	4.4	29	5.1
	Metal work	302	4.4	28	5.1
	Demolition	315	4.5	13	3.8
	Radiator repair	303	4.4	26	5.4
	Plumbing	310	4.5	18	4.1
	Sandblasting	317	4.4	12	5.4
	Auto body work	294	4.4	35	5.1
	Painting	273	4.4	56	4.7
	Welding	295	4.3	34	***5.7***
	Other lead handling	308	4.7	17	5.0
HOBBIES INVOLVING LEAD		225	4.4	2	4.5
SOCIO-ECONOMIC FACTORS	Mother is homemaker	169	4.2	162	***4.8***
	Mother's education more than high school	168	4.5	156	4.6
	Father's education more than high school	154	4.5	98	4.6
	Income less than $15,000	218	4.4	96	4.8

Numbersin bold/italics indicate that the mean among the exposed is statistically significantly greater than the mean among the enexposed ($p<0.05$).

Considering the large number of factors analyzed, several variables are expected to be statistically significant just by chance. Even with respect to the few statistically significant variables, the actual difference between mean levels was less than 1µg/dL. The largest difference in lead levels seemed to be related to the laboratory where the analysis was performed.

Our study was unable to identify any new risk factors for high blood lead levels.

Other possible explanations for the low blood lead levels found in this study are: Low soil and air levels due to steady circulation of fresh air, relatively small percentage of homes built before 1950, good maintenance of old houses (many of them are located in neighborhoods with high property prices), high percentage of time spent outdoors and the comparatively good nutrition status of children in Hawaii. The low levels in Maui may be a result of selection bias: All children on Maui were recruited through private physicians and only venous blood samples were taken. Another explanation could be the higher proportion of newer housing on Maui; 32% of all housing on Maui was built since 1980, as opposed to 21% overall in the state. Only 19% of housing on Maui was built before 1950 as compared to 27% in the state (Census Data 1990).

Conclusions

1. The mean blood lead level of 389 children was found to be 4.5µg/dL and none had levels above 14µg/dL. If more children had been tested, a few children with levels above 14µg/dL might have been identified.
2. The level of parental knowledge and concern about lead and by health professionals varies widely. Overall it appears to be rather low. In combination with a widespread reluctance to subject a child to a phlebotomy, it resulted in a low participation rate in blood lead testing.
3. Children living in different geographic areas and housing types, with varying socioeconomic and educational backgrounds, were included in our study. Thus, despite the high refusal rate, there is no reason to think that the true distribution of blood lead levels among children in Hawaii differs drastically from our results.
4. Based on our study, mandatory lead testing of all children in Hawaii is not recommended. However, health care providers should routinely inquire about risk factors, such as flaking paint in old houses, parents' occupational exposure to lead, rainwater catchment systems, toys and hobbies that might be contaminated by lead. Children exposed to any one risk factor should be tested. However, living in a house older than 30 years cannot be regarded as a risk factor unless flaking or chipping paint is present.
5. Public education dealing with sources and risks of lead is essential in preventing exposure to lead in the future and in achieving an appropriate level of diagnostic suspicion.

ACKNOWLEDGMENTS

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EDITORIAL
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Ma'i Ulu

In this issue of the *Journal*, we recognize our neighbors to the south—Amerika Samoa.

American Samoans have the rights of citizenship in the USA. Our community includes a great many Samoans and Samoan athletes have put Hawaii on the world map. The coming and going between here and Samoa fill the airplanes.

It seems quite appropriate, therefore, that we have a research article on a public health issue in Amerika Samoa—"Stroke and Traumatic Brain Injury in that southern Pacific group of the islands—known to Samoans as *Ma'i Ulu*."

The author, Gloriajean L Wallace PhD, researched extensively on the subject while she was based in Hawaii at the University during the last decade. She is a speech-language pathologist with imposing credentials and has had a particular interest in the health and well-being of the Samoan people.

J I Frederick Reppun MD

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